

## Acute kidney injury: The crossroads of ICD-9-CM and medical literature

HCPPro

*Association of Clinical Documentation Improvement Specialists*, September 1, 2010

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In severity and risk adjustment, defining acute kidney disease, specifically acute renal failure (ARF) and its synonym, acute kidney injury (AKI), are confounding issues for physicians, coders, and quality specialists. When do patients with elevated creatinine levels or oliguria have ARF or AKI, a major complication and comorbidity under MS-DRGs? How do ARF, AKI, and other terms, such as acute renal insufficiency or azotemia, factor in risk adjustment?

### **AKI's prevalence and impact**

Depending on the definition, AKI occurs in 4.9%– 7% of inpatient admissions. A 2009 paper in the *Journal of the American Society of Nephrology*(<http://tinyurl.com/AKI-Mortality>) reports that 90-day mortality increases in proportion to the AKI stage. Even so, many physicians specify significant creatinine elevations as prerenal, renal, or postrenal azotemia (code 790.6, other abnormal blood chemistry) or acute renal insufficiency/dysfunction (code 593.9, unspecified disorder of kidney and ureter).

The above codes have little, if any, effect in risk adjustment methodologies measuring clinical performance. On the other hand, ARF or AKI codes (even without dialysis) reflect greater resource consumption and mortality if defined, diagnosed, documented, and, most importantly, associated with their suspected underlying renal pathology. ARF or AKI codes without a specified renal pathology, such as 584.9 (ARF, unspecified) are weighted less than those with specified pathology; thus, the need to define and diagnose their suspected underlying renal pathology (e.g., acute interstitial nephritis, acute tubular necrosis) is critical.

### **Defining AKI**

Physicians must identify AKI to code it properly. This is confusing because two prevailing definitions of AKI and ARF exist.

- The Acute Kidney Injury Network (AKIN) definition may be viewed at <http://ccforum.com/content/11/2/R31>
- The Acute Dialysis Quality Initiative Group (ADQIG) RIFLE criteria may be viewed at <http://ccforum.com/content/8/4/R204>.

These definitions differ in some respects. The AKIN definition stipulates that volume depletion has been corrected when measuring the serum creatinine change, whereas RIFLE does not. Although an acute creatinine rise of 150% qualifies for AKI in the AKIN criteria, it does not in RIFLE. Read these references carefully to properly apply and defend them in your medical decision-making and subsequent documentation.

Note that fractional excretion of sodium, urine osmolality, presence of granular casts of microscopic urinalysis, or need for dialysis are not defining parameters. These should be documented, however, given that they add credibility to the presence of renal pathology and that no reliable defining AKI biomarker (e.g., urine NGAL, serum cystatin-C) exists.

## **Need for suspected renal pathology diagnosis**

Although most physicians document the instigating cause of AKI (e.g., hypovolemia, medications, sepsis, ureteral obstruction), they fail to document the suspected renal pathology, given that they do not usually perform renal biopsy.

Most of us have a clinical sense of the renal pathology in AKI. As we know, medications can impair glomerular function (e.g., NSAIDs) or cause acute tubular necrosis (e.g., aminoglycosides), acute interstitial nephritis (e.g., phenytoin), glomerulonephritis (e.g., ampicillin), or osmotic nephrosis (e.g., IVIG). Prolonged hypovolemia, DIC, or shock can cause ischemic acute tubular necrosis (ATN), whereas myoglobin causes a nephrotoxic ATN. Interstitial nephritis can result from bilateral pyelonephritis while bilateral cortical necrosis can result from preeclampsia. Documenting these suspected pathologies, especially at the time of discharge, demonstrates higher medical decision-making and illness severity than AKI alone.

Limitations of ICD-9-CM Coding is based on explicit physician documentation, especially at the time of discharge. The abbreviation of AKI cannot be coded as acute kidney injury unless it has been written out at least once. Unfortunately, ICD-9-CM does not specify the different AKI stages (e.g., AKIN Stage 1–3); thus, ICD-9-CM databases cannot distinguish the more severe cases without clinical abstraction.

Physicians must document consequences, such as fluid overload or metabolic acidosis, for coders to report them. In summary, physician identification of and specificity in defining AKI using ICD-9-CM terminology better reflects severity of illness and mortality risk. Until nephrologists and critical care physicians clearly establish applicable diagnostic criteria—and ICD-9-CM follows suit by allowing for AKI staging—we must document its suspected underlying cause and consequences.

Reserve azotemia or “acute renal insufficiency” for patients who do not meet AKI criteria.

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